

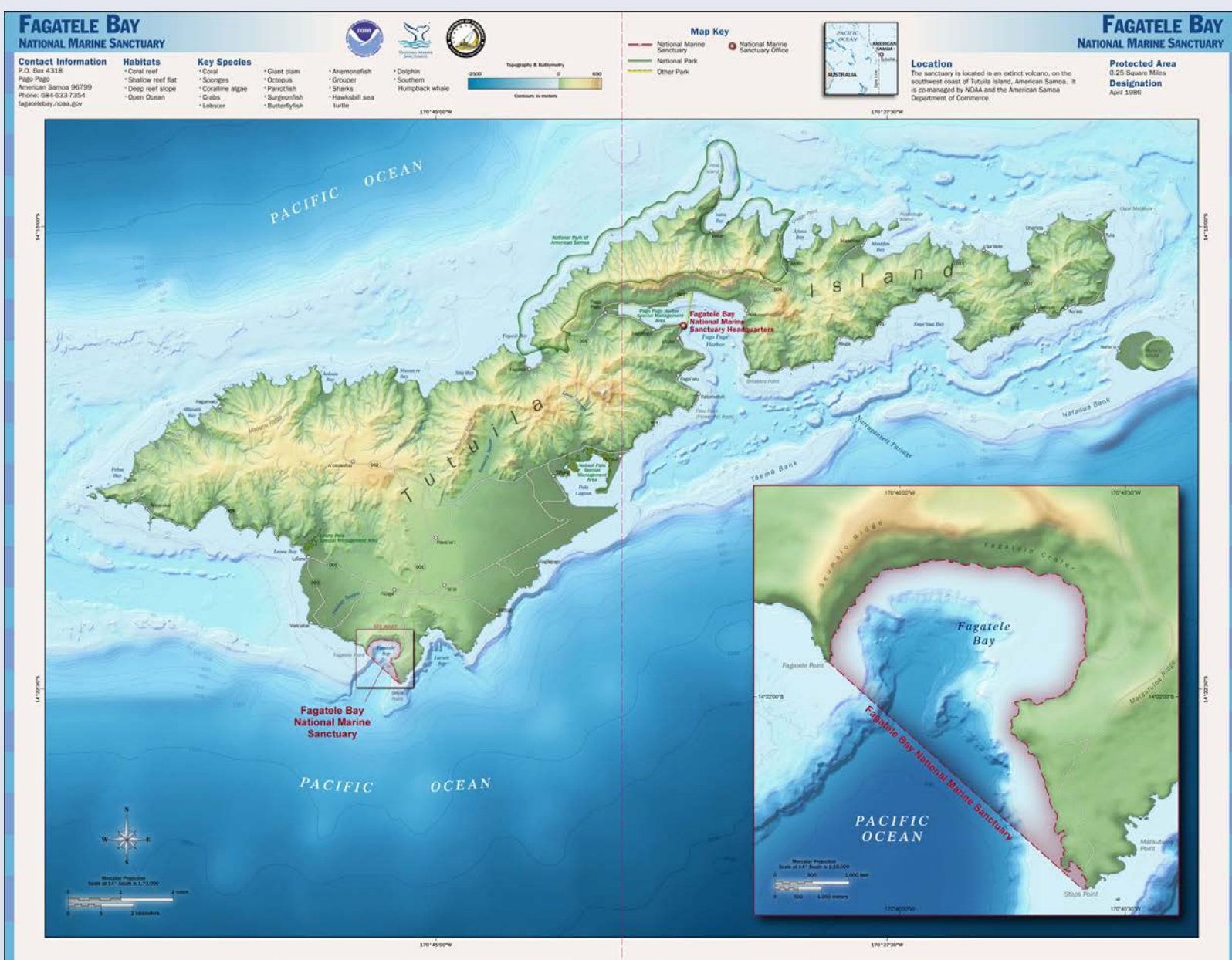
Allostatic Load Varies by Apolipoprotein E and Ace Genotypes in American Samoans

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INTRODUCTION

- Allostatic load (AL) manifests from both failed and successful morphological and biological stress responses across physiological systems.
- Genes, culture, and environment affect responses to stressors.
- AL is enhanced by senescent processes, predisposes individuals to chronic non-communicable diseases, and predicts future morbidity and mortality (Crews, 2007).
- This measure represents physical decline from “optimal” physiological function to a more dysfunctional phenotype.
- Reported research has yet to explore associations of genetic markers with AL: for this reason we examined associations of apolipoproteins E and H, Angiotensinogen Converting Enzyme (ACE), and Atrial Natriotic Peptide (ANP) with AL.



Source: www.en.wikipedia.org

HYPOTHESES

Hypotheses were formed based on the idea that certain genes can influence predisposition to higher allostatic load. Based on this assumption, we utilized the following hypotheses:

- Individuals with the apolipoprotein E (3,2) genotype will have significantly lower AL than individuals with the (2,2) or (3,3) genotypes.
- Individuals who are homozygous dominant for apolipoprotein H (2,2) will have significantly lower AL than individuals with the (2,1) genotype.
- Individuals with the ACE (I-D; insertion-deletion) genotype will have significantly lower AL than individuals with the ACE (I-I; insertion-insertion) genotype.
- Individuals with the ANP(TT; top-top) genotype will have significantly lower AL than individuals with the (TB; top-bottom) or (BB) genotypes.

METHODS

- Physiological and morphological variation among 284 American Samoans along with blood samples were obtained in 1989 (Crews et al 1991, Crews 2007).
- These data included multiple secondary mediators of allostasis.
- Here we examine associations between 3 constructs of AL and genotypes at the apolipoprotein E and H, ACE, and ANP loci.

Allostatic Load 1: Secondary Mediators

Systolic blood pressure > 156 = AL + 1
Diastolic blood pressure > 92 = AL + 1
Total cholesterol > 222 = AL + 1
Waist-hip ratio > 91.5 = AL + 1
Glycated hemoglobin > 1060 = AL + 1
HDL cholesterol < 24.6 = AL + 1
Fasting serum insulin > 214 = AL + 1

Allostatic Load 2: Body Habitus

Total cholesterol > 222 = AL2 + 1
HDL cholesterol < 24.6 = AL2 + 1
Waist-hip ratio > 91.5 = AL2 + 1
BMI > 33.1 = AL2 + 1
Systolic blood pressure > 156 = AL2 + 1
Diastolic blood pressure > 92 = AL2 + 1
Glycated hemoglobin > 1060 = AL2 + 1
Fasting serum insulin > 214 = AL2 + 1
Triceps skinfold > 55.3 = AL2 + 1
Subscapular skinfold > 60 = AL2 + 1
Relative fat pattern index > .50 = AL2 + 1

Allostatic Load 3: Glucose and Lipids

Total cholesterol > 222 = AL3 + 1
HDL Cholesterol < 24.6 = AL3 + 1
LDL cholesterol > 163 = AL3 + 1
Triglycerides > 137 = AL3 + 1
Waist-hip ratio > 91.5 = AL3 + 1
Systolic blood pressure > 156 = AL3 + 1
Diastolic blood pressure > 92 = AL3 + 1
Glycated hemoglobin > 1060 = AL3 + 1
Fasting serum insulin > 214 = AL3 + 1
Fasting serum glucose > 179 = AL3 + 1

Using SPSS/PC+ Statistical Package for IBM PC, we performed regression analyses and determined variance and significant relationships between the genotypes and AL ($p < .05$ for all measures).

RESULTS

- Significant relationships were determined for associations between AL and apolipoprotein E and ACE genotypes.
- Persons with the apolipoprotein E (3,2) genotype showed significantly lower AL than the (3,3) or (2,2) genotypes ($p < .05$).

Comparisons of F-values and significance between groups for apolipoprotein E with ALOAD, ALOAD2, and ALOAD3 among 283 American Samoans.

	F	Sig.
ALOAD	3.7479	.0251
ALOAD2	1.0631	.3471
ALOAD3	5.2850	.0057

- Persons with the ACE (I-D) genotype showed significantly lower AL than persons with the ACE (I-I) genotype ($p < .05$).

Comparisons of F-values and significance between groups for ACE with ALOAD, ALOAD2, and ALOAD3 among 283 American Samoans.

	F	Sig.
ALOAD	4.7243	.0306
ALOAD2	2.3990	.1226
ALOAD3	4.6472	.0320

- Nonsignificant relationships were observed between AL and apolipoprotein H and ANP genotypes.

Comparisons of F-values and significance between groups for apolipoprotein H with ALOAD, ALOAD2, and ALOAD3 among 283 American Samoans.

	F	Sig.
ALOAD	.4213	.5169
ALOAD2	.0951	.7580
ALOAD3	.6692	.4141

Comparisons of F-values and significance between groups for ANP with ALOAD, ALOAD2, and ALOAD3 among 283 American Samoans.

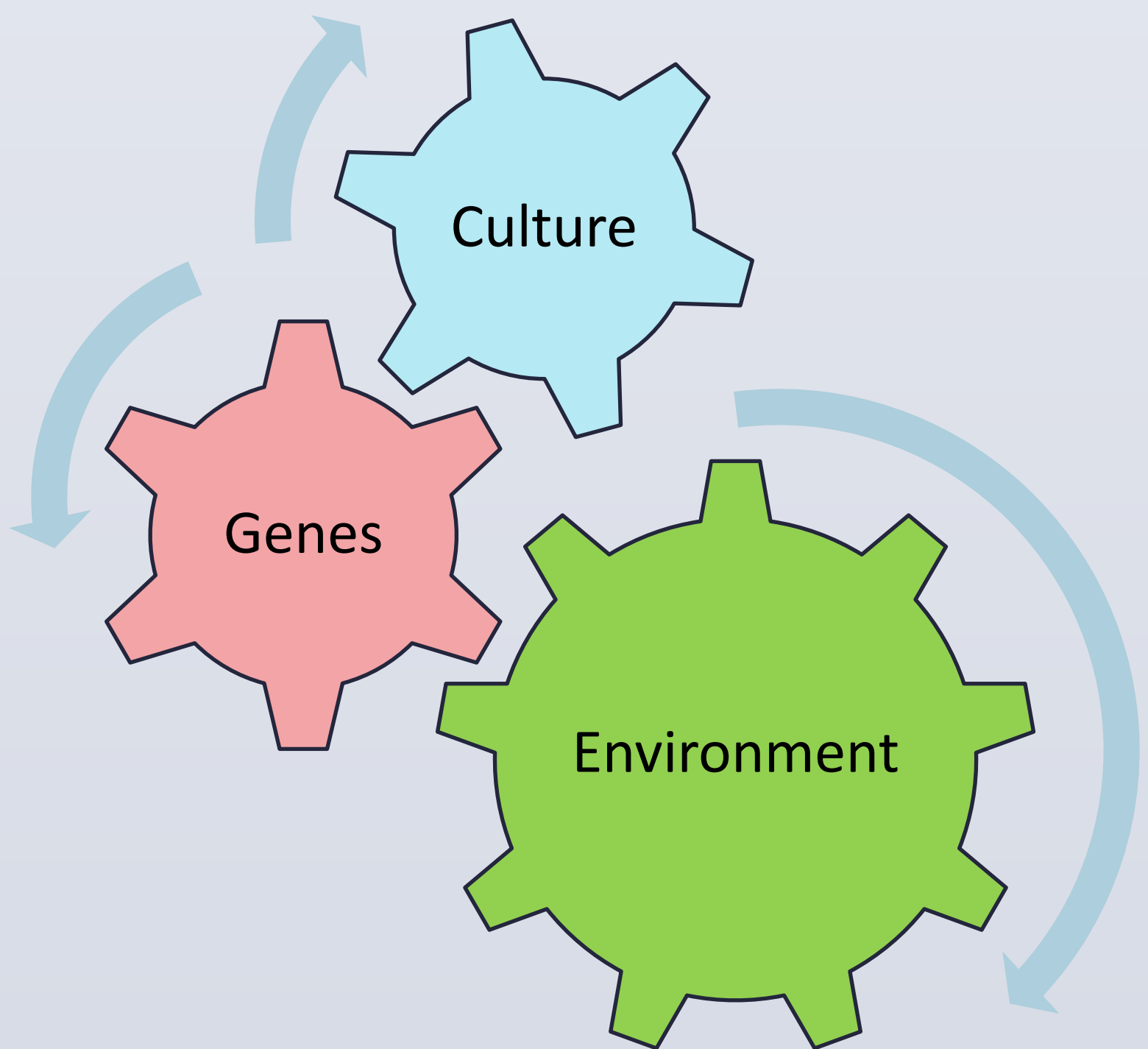
	F	Sig.
ALOAD	.0127	.9874
ALOAD2	.0329	.9676
ALOAD3	.1406	.8689

CONCLUSIONS

Genetic predictors of physiological dysfunction may be important modulators of risks for morbidity, senescent biology, and mortality across populations. Apolipoprotein genes have high predictive power of chronic disease risk, including coronary heart disease (Benderly et al, 2009). Thus, examination of associations between AL and apolipoprotein E and ACE genotypes is crucial for understanding effects of genes on chronic stress.

FUTURE ANALYSES

Because relationships between genes and allostatic load have only recently been examined in scientific literature, many more analyses need to be conducted. These analyses should include examinations of AL/genotype associations across non-Samoan populations to account for population differences in genetic risk for high AL. Dr. Crews and I are currently examining interactions between AL, genotype, age, sex, and education. Future analyses should include additional social variables.



REFERENCES

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